Amendments to the Specification

Please replace the first paragraph on page 1 with the following paragraph:

--This application is a continuation-in-part of U.S. Patent Application Serial No. 09/496,771 filed on February 3, 2000, now issued as U.S. Patent 6,355,271,which claims benefit of the filing dates of U.S. Provisional Application Serial Nos. 60/118,356; 60/118,364; and 60/118,355, all filed February 3, 1999, the entire contents of each of which are hereby incorporated by reference. This application also claims priority to U.S. Provisional Application No. 60/267,357 filed on February 8, 2001, entitled "Casein-Complexation of Calcium Phosphate Particles Containing Insulin as Oral Delivery System," the entire contents of which are hereby incorporated by reference.--

Please replace the second paragraph on page 2 with the following paragraph:

--For example, diabetes mellitus is a metabolic disease in which there is a deficiency or absence of insulin secretion by the pancreas. It is characterized by hyperglycemia, glycosuria, and alterations of protein and fat metabolism, producing polyuria, polydipsia, weight loss, ketosis, acidosis, and coma. See GOULD'S MEDICAL DICTIONARY, 381 4th ed. 1979. Diabetes mellitus is often inherited, but it may be acquired. The disease occurs in two major forms: Type I, or insulin-dependent diabetes mellitus, and Type II, non-insulin-dependent diabetes mellitus. The condition may also be gestational (Type III), or due to impaired glucose tolerance (Type V). Type IV encompasses all other forms of diabetes, including those that are associated with pancreatic disease, hormonal changes, adverse effects of drugs, or genetic or other anomalies. ~~See www.harcourt.com/dictionary/def/2/9/4/9/2949900.html.--~~

Please replace the third paragraph on page 2 with the following paragraph:

--Specifically, Diabetes, Type I is an insulin-dependent diabetes (IDDM), now known to be a T-cell mediated autoimmune disease that specifically targets the pancreatic ß-cells. It causes a deficiency strongly correlated to a hereditary predisposition to injury or destruction of pancreatic ß-cells, which produce and secrete insulin. The ß-cell insufficiency and destruction is

generally caused by chemical-pH imbalances and viral or antibody damage, such as that caused by inflammatory cytokines, particularly those produced by TH1-type lymphocytes, which are hypothesized to play a major role in the pathogenesis of all autoimmune diseases, including diabetes of this type. Individuals are susceptible to Type I at an early age and usually suffer childhood onset. ~~See http://vaxa.com/html/669.cfm.~~

Please replace the first paragraph on page 3 with the following paragraph:

--Diabetes, Type II is a non-insulin dependent diabetes (NIDDM), being a disorder of glucose homeostasis characterized by hyperglycemia, peripheral insulin resistance, impaired hepatic glucose metabolism, and diminished glucose-dependent secretion of insulin from pancreatic ß-cells. This latter defect may lie in the glucose signaling pathway in ß-cells involving metabolically regulated potassium channels, which are the targets of sulphonylurea drugs commonly used in the treatment of NIDDM. Type II is characterized by insulin insensitivity, which is typically evidenced by high levels of circulating insulin and the reversibility of blood sugar elevation (by dietary changes and/or weight loss), sufficient to restore insulin sensitivity. Low GTF chromium levels are a major determinant of insulin insensitivity; obesity is another significant factor. Onset of Type II is generally diet related and usually occurs later in life. ~~See id.~~ --